# A Suicide Attempt Using Zinc Phosphide (A Case Study)

# Zinc Phosphide ile Meydana Gelen Bir Suisid Vakası (Olgu Sunumu)

Zinc Phosphide ile Suisid Girişimi / Suicide Attempt Using Zinc Phosphide

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**Özet**

Çinko Fosfür, fare mücadelesinde zehirli buğday üretimi için kullanılır ve rodentisitin aktif maddesidir. İntihar amacıyla tarım ilacı [Zinc Phosphide (Zn3P2)] içen 17 yaşındaki erkek olgu acil serviste görüldüğünde: Genel durumu kötü, bilinci kapalı, kusmuğu ve cildinde sarımsak kokusu ve ileri derecece asidozu mevcuttu. Semptomatik tedavi uygulanan ve mekanik ventilatorde takip edilen olgu yatışının 5. günü psikiyatri kliniğine devir edildi.

**Anahtar Kelimeler**

Çinko Fosfür; Suisid; Sarımsak Kokusu

**Abstract**

Zinc phosphide is a toxin that is added to wheat for use in rodent control and is the active ingredient of rodenticide. A 17 year-old male attempted suicide by drinking pesticide [Zinc PHOSPHIDE (Zn3P2)] and was subsequently admitted to the emergency department: the patient's general condition was poor, he was unconscious and vomiting, the skin had a garlic odor and advanced acidosis was present. The patient was treated symptomatically, followed by mechanical ventilation, and was transferred to a psychiatric clinic on the fifth day.

**Keywords**

Zinc Phosphide; Suicide; Smell of Garlic

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## Introduction

Organophosphate poisoning is a significant cause of morbidity and mortality in young and economically active age groups from developing countries all over the world, , particularly those of low educational and socio-economic status. Zinc phosphide, the active material of rodenticide, is used in the production of toxic wheat for rodent control. Zinc phosphide Minimum 80% of zinc phosphide involves 19% active phosphorus. Zinc phosphide is a black, heavy powder. It is largely insoluble in water and has an odor like garlic or carbide, which the smell of the phosphorated hydrogen gas (PH3 , phosphine) emerging after dissolution upon meeting a gas. Both history of exposition and the smell of garlic or rotten fish help diagnosis. The lethal dose of zinc phosphide is 20- 40 mg/kg for humans [1].

We present the case of a 17 year-old male who attempted suicide by drinking pesticide [Zinc Phosphide (Zn3P2)].

## Case Report

When the patient was observed in the emergency department; general condition was poor, he was unconscious, intubated, was unresponsive to painful stimulus, in GCS:3, papillary was advanced miotic (pin point), light response was +/+, respiratory sound was coarse with increased secretion. Relatives of patients given the drug by the time and dose was unclear zinc phosphide. The case was vomiting and had a garlic smell from his skin, and was isolated for 48 hours to prevent the poisoning of other patients. His noninvasive blood pressure was 80/40 mmHg, and pulse was 94/min. Toxicological information sources were consulted. The patient was admitted to the intensive care unit to prevent the progression of metabolic acidosis, hypo/hyperglycemia, ventricular dysrhythmia, hypotension, tachycardia, ataxia, hepatotoxicity effect, acute renal failure. Analysis of arterial blood gas showed that pH was 7.11, PaO2 as 44 mmHg, HCO3 as 14.6 mmol/L and SpO2 as 85%. Propofol infusion was commenced and followed as FiO2:0.5, Vt:6 mL/kg, frequency:14/minutes, and PEEP:5 cmH2O in SIMV mode in a mechanical ventilator. The patient was given 2mg/kg activated carbon (Char-Flo) with a naso-gastric catheter. 2000cc fluid per day was given to the patient, urine output was observed as normal. Liver function tests and other biochemical parameters remained normal values. Hemodynamics remained stable. Additional drug treatment did not implemented. The case did not require atropine. The patient's papillary healed after 6 hours. Further analysis of blood gas showed that pH:7.38, PaO2:87 mmHg, PaCO2:34mmHg, HCO3:21 mmol/L, SpO2: 95%. Morning sedation was ceased and the patient was extubated on the second day. Additive pathology was not detected in tracks. The patient was transferred to a psychiatry clinic on the fifth day when he did not require further intensive care.

## Discussion

In the mechanism of zinc phosphide poisoning: poisoning is mediated by phosphine, which was thought to act by inhibiting cytochrome C oxidase. While phosphine does inhibit cytochrome C oxidase in vitro, the inhibition is much less in vivo. It was recently shown in nematodes that phosphine rapidly perturbs mitochondrial morphology, inhibits oxidative respiration by 70%, and causes a severe drop in mitochondrial membrane potential. This failure of cellular respiration is likely to be due to a mechanism other than inhibition of cytochrome C oxidase. In addition, phosphine and hydrogen peroxide can interact to form the highly reactive hydroxyl radical and phosphine also inhibits catalase and peroxidase; both mechanisms result in hydroxyl radical-associated damage such as lipid peroxidation. The major lethal consequence of phosphide ingestion, profound circulatory collapse, is secondary to factors including direct effects on cardiac myocytes, fluid loss, and adrenal gland damage. In addition, phosphine and phosphides have corrosive actions [2]. Zinc phosphide is a crystalline powder with an odor resembling rotten fish. It is available as a powder or as pellets that release phosphine gas on contact with water. The clinical features of zinc phosphide poisoning are similar to those of aluminium phosphide but slower in onset, since the release of phosphine is slower. Nausea and vomiting are early features and can occur after ingestion of as little as 30 mg. Chugh et al. [3] reported that patients complain of tightness in the chest and may be excited, agitated and thirsty. Shock, oliguria, coma and convulsions may develop. Pulmonary edema, metabolic acidosis, hypocalcaemia, hepatotoxicity, thrombocytopenia and ECG changes are seen. The management of zinc phosphide poisoning is mostly supportive and symptomatic. Stomach irrigation and activated carbon applications are efficient within the first hour. Saleki et al. [4] stated that PH3 can cause liver dysfunction, especially after the first day of poisoning, and that the main histopathologic changes found were fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. A similar condition was recorded in a case reported by Khurana et al. [5], who found elevated transaminase levels; Karanth and Nayyar [6] recorded severe hepatic dysfunction in their cases. Musshoff et al. [7] found congestion, edema and centrizonal necrosis of the liver from histopathological examination.

Chugh et al. [3] reported that zinc phosphide (Zn3P2) poisoning was rare before 1986, but that twenty cases of ingestion (self-poisoning) were seen during the five years preceding the study (January 1992-December 1996). Profuse vomiting (100%), abdominal pain (100%), palpitation and sweating (80%), dyspnea and tachypnea (75%), metabolic acidosis (60%), shock (40%), and hypotension (40%) were the most common presenting features. Five patients (25%) died. The toxic effects were due to liberation of toxic phosphine (PH3) gas which was detected by qualitative silver nitrate paper test in the majority of cases.

Orak et al. [8] reported the diagnosis of acute distal renal tubular acidosis (type 1) and deep metabolic acidosis (ph:6.973, PaCO2 24.2 mm Hg, PaO2 91 mm Hg, and HCO3 9.3 mEq/L, lactate 2 mEq/L) in a 25 year-old zinc phosphide poisoned male patient. They recommended that respiratory support and intravenous fluid resuscitation with sodium bicarbonate treatment should be considered.

Vomiting should be avoided, as patients develop toxic phosphine gas in the stomach upon taking zinc phosphide orally. In case of vomiting, attention should be paid to prevent secondary contamination within enclosed areas.

### *Competing interests*

The authors declare that they have no competing interests.

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